are: East Anglia 12 inches, south-west England 14 inches and Wales 23 inches.

Of 203 cases available for us to study from clinical case-notes, occupation was not recorded in 23. Of the remaining 180, one was a tilemaker, one was a jobbing gardener, and the other 178 were farmers, agricultural or horticultural workers, millers, seedsmen, or employed in the repair and maintenance of farm buildings. There were 5 whose agricultural occupation was part-time (the 'second job' that helps the family budget). Outside of our survey figures, I have knowledge of the disease occurring in a schoolteacher and an elderly woman of independent means, both of whom were keeping, and feeding, agricultural animals.

The dusts that caused the illness in our cases were of hay (60%), hay and straw together (28%), straw crops (7%) and miscellaneous dusts (5%). The jobs of work from which onset was dated were: Cattle feeding and allied haywork, including baling and unbaling of stored hay (56%), threshing (30%), work with threshed corn and grains (6%) and miscellaneous (8%).

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Atypical Interstitial Pneumonia of Cattle

Blood (1962) proposed the term atypical interstitial pneumonia to cover a clinical syndrome in cattle known for many years under a variety of names, including panters, lungers, fog fever and pulmonary adenomatosis. In this country the syndrome is generally termed fog fever, fog denoting pasture grazing after the removal of a crop of hay or grass. The fog fever syndrome is characterized by a sudden and violent attack of acute respiratory distress in groups of animals sometimes accompanied by subcutaneous emphysema; the mortality rate is high. Occasional outbreaks of a more chronic nature occur with milder clinical signs including coughing, depression of appetite, loss of weight, and reduction of milk yield in dairy cattle; recovery in such outbreaks is slow. The syndrome is seldom seen in animals under 3–4 months of age and, although it may occur at any time of the year, workers both in this country and North America have reported a marked seasonal occurrence in the late summer and autumn (Barker 1948, Begg & Whiteford 1948, Blood 1962). More recently, the acute form of the syndrome has been met in young animals of 6–12 months of age reared under intensive indoor beef production systems. Frequently in these outbreaks a history can be obtained of mild respiratory disease earlier in life with apparent uneventful recovery. In the absence of convincing experimental observations, the ætiology of the syndrome remains obscure. Jarrett *et al.* (1953) suggested that the severe respiratory clinical signs could result from an increased respiratory rate associated with an obliteration of a large volume of pulmonary air space. Michel (1954) produced clinical signs resembling the fog fever syndrome in young cattle artificially infected with lung-worm larvæ; respiratory distress occurred when a massive dose of infective larvæ was given to an animal that had recently recovered from a previous artificial infection. The general consensus of opinion would indicate that allergy plays some part in precipitating the syndrome and various allergens, in addition to lung-worm larvæ, have been suggested. They include grass protein (Barker 1948) and hay dust (Blood 1962). Gregory & Lacey (1963) examined samples of hay associated with farmer's lung, and in contrast to samples of both normal and mouldy hay, these samples were rich in thermophilic organisms with hundreds of millions of actinomycetes spores per gram of hay. Many spores were shown potentially able to penetrate to the deeper parts of the lung especially those of actinomycetes, *Aspergillus fumigatus* and *Hemispora stellata*. The role of such agents in the ætiology of atypical interstitial pneumonia of cattle requires further investigation.

REFERENCES
Barker J R (1948) *Rep. Proc. Conf. Grassl. & anim. Hlth*, London, Oct. 28–29. N.V.M.A. publ. No. 17, p 86
Begg H & Whiteford W A (1948) *Vet. Rec.* **60**, 135
Blood D C (1962) *Canad. vet. J.* **3**, 40
Gregory P H & Lacey M E (1963) *J. gen. Microbiol.* **30**, 75
Jarrett W F H, McIntyre W I M & Urquhart G M (1953) *Vet. Rec.* **65**, 153
Michel J F (1954) *Vet. Rec.* **66**, 381

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The Syndrome of 'Broken Wind' in the Horse

'Broken wind' in the horse is characterized by a diphasic expiratory dyspnœa. The first normal and passive expiratory movement is supplemented by an active and forceful contraction of the abdominal musculature to expel the remaining tidal

air. Defined ætiologically the term 'broken wind' implies a generalized constriction of the smooth muscle of the bronchioles and alveolar ducts, possibly of allergic origin. Classified in terms of pathology the end product of this obstruction to expiration is a generalized pulmonary alveolar emphysema. We recognize a *functional* alveolar emphysema which is potentially reversible and in which the alveoli appear normal at post-mortem examination; and a *structural* emphysema which is permanent and in which the alveolar walls are ruptured (Arnott 1956). Physiologically the condition is characterized by a positive intrapleural pressure on expiration (Obel & Schmiterlöw 1948).

The Clinical Picture

An acute and a chronic form may be described. The *acute* form shows a sudden and dramatic onset of severe, expiratory dyspnœa, analogous in many ways to the acute type of human asthma, the attack generally coinciding with a change of environment. In our experience this form is particularly likely to occur in thoroughbred brood mares during the summer when, after spending the day in a field, they are brought in to be stabled overnight. The attack may commence within twenty minutes of being stabled. The affected animal develops prominent abdominal expiration, flaring of the nostrils and a rhythmic movement of the anus and perineum which coincides with respiration. Restlessness, profuse or patchy sweating and a tendency to resent handling may also be noticed. Paroxysmal coughing, with a deep, frequent and non-productive cough, is accompanied by loud expiratory 'wheezing' clearly audible without the use of a stethoscope. Auscultation of the chest reveals sounds which are similar in type, though more exaggerated, to those that will be described under the chronic form. The pulse may be full and frequent, but there is no significant pyrexia. These attacks are not fatal but may last for several hours and may be followed by daily episodes of similar severity if the management is unaltered. Subsequently the case will progress insidiously into the chronic form.

Unless the acute form precedes, the *chronically* emphysematous horse may have a history of mild respiratory abnormality for some months or even years before veterinary attention is sought. A typical history would be of the horse which has been heard to cough occasionally in the stable or when first walked out from a warm stable into the cold air and given a sharp trot. Examination of the horse at rest shows an increased respiratory rate with a prolonged and diphasic expiration. In a mild case this may be the only physical abnormality, auscultation of the chest and trachea revealing no marked deviation from the normal. Periodic exacerbations of this quiescent state are, however, a marked feature of chronic emphysema. These exacerbations may be associated either with the development of a chronic bronchitis, with increased coughing, a slight nasal discharge and mucous rales on auscultation, or with the flare up of an allergic state. As the disease progresses and especially if the degree of chronic bronchitis is marked, a 'wheezing' sound becomes audible on auscultation in the second forced expiratory phase. These rhonchi may also be accompanied by mucous rales on both expiration and inspiration. Percussion of the chest, in both acute and chronic emphysema, will reveal that the lung is abnormally distended (Carlström & Alegren 1940).

Exercise tolerance is reduced and advanced cases become 'barrel chested' and unfit for anything except the lightest work. Congestion of the pulmonary circulation and a tendency to cor pulmonale does occur (Salutini 1959). Cachexia may be seen in advanced cases but initially the chronically emphysematous horse may be a greedy feeder and therefore in rather gross condition.

Diagnosis

Diagnosis presents no particular difficulty in that the symptoms are characteristic and conditions which simulate generalized emphysema are uncommon. Pulmonary tuberculosis and neoplasia, both of which are comparatively rare, should be borne in mind. Persistent coughing may be caused by a lung-worm infestation and in these cases eggs or larvæ can be detected by fæcal examination (Fletcher 1960).

Prognosis

By definition, *structural* emphysema is irreversible and tends to be progressive. The prognosis is therefore unfavourable, though with careful management the horse may be fit for light work for many years. The prognosis for *functional* emphysema is only a little better, in that most cases would seem to progress into the structural form and even those that appear to make a recovery probably remain 'sensitized'. The prognosis is considerably worsened for both forms by anything that precipitates the allergic state (e.g. atmospheric pollution with mould or dust) or by conditions of management which aggravate bronchial catarrh (e.g. poor ventilation, respiratory infections).

Treatment

Functional emphysema is potentially curable but structural emphysema is not. However, a certain amount can be done to alleviate the symptoms of both forms. The most beneficial therapy is *fresh air*. Ideally the horse should be kept 'out at grass', but if the horse must be stabled, measures should be taken to reduce stable dust to a minimum. The feeding of a small quantity of well damped, good quality hay and the replacement of the straw bedding by peat moss or wood shavings are important factors in arresting the disease.

As a supplement to the 'clean air' treatment certain medicinal therapies are helpful. Temporary relief of the acute syndrome can be achieved by the use of corticosteroids, atropine and adrenaline. Antibiotic therapy and expectorants may help to reduce the respiratory distress caused by a concurrent chronic bronchitis.

Morbid Anatomy

We know of no post-mortem evidence from horses destroyed during an acute attack, but recently we had the opportunity to study material from two thoroughbred brood mares that had suffered a series of acute emphysematous attacks during the three months before euthanasia and which on post-mortem evidence were judged to be suffering from *functional* emphysema. Macroscopically the lungs revealed no deviation from normal.

As compared with normal equine lung the microscopic appearance of sections taken from these cases showed the following features:

(1) An alveolar pattern and wall structure compatible with normal.

(2) A peribronchiole infiltration with monocytes, lymphocytes, plasma cells and occasional eosinophils.

(3) A marked increase in the mucus-secreting cells of the bronchi (Fig 1).

The lung pathology in *structural* emphysema is said to be similar (Stommer 1887), if not identical (McLaughlin *et al.* 1961*a*, *b*), to the condition in man. Macroscopically one sees the typical uncollapsed pale, dry, 'air-cushion' type of lung with subpleural bullæ along the apices and periphery.

Microscopically the alveolar pattern is disrupted and there is a thinning of the alveolar walls (Fig 2A). The peribronchiolitis described in the functional form is more severe and a marked fibrosis extends towards the alveoli, obliterating the air spaces (Fig 2B). In many cases, though by no means all, there is a chronic bronchitis (Fig 2C) and the lumen of the bronchi are filled with 'plugs' of mucopus.

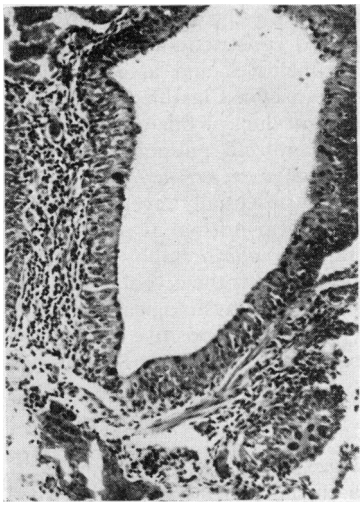

Fig 1 *A section showing the peribronchiolar infiltration with round cells and the prominent and numerous mucus-secreting cells of the bronchioles. H & E.* ×80

Ætiology

Many hypotheses have been proposed regarding the ætiology of emphysema in the horse, since the year 333 B.C., when a disease characterized by 'a drawing in of the flank' was described by Aristotle (Smith 1919). On the evidence available – and it is surprisingly scanty – an allergic ætiology seems probable. It is surely significant that on one point there is *universal* agreement, that the feeding of dusty or mouldy hay is harmful.

Emphysema in the horse has been described as 'a histamine susceptible condition' (Obel & Schmiterlöw 1948). Emphysematous horses show an increased sensitivity to the injection of histamine, as do human asthmatics (Weiss *et al.* 1929). The intravenous injection of histamine in a normal horse produces a syndrome which, except for the absence of cough, is almost identical to that already described under the heading of acute emphysema (Andberg *et al.* 1941). There is disagreement on the question of the blood histamine level in the naturally occurring disease. Zanzucchi (1942) reported that chronically emphysematous horses had twice the normal blood histamine level, but Brion *et al.* (1948) failed to confirm this and Code & Hester (1939) reported that anaphylactic reactions in the horse do not cause a rise of the blood histamine level.

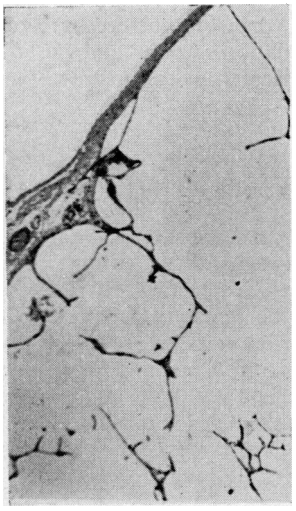

Fig 2A

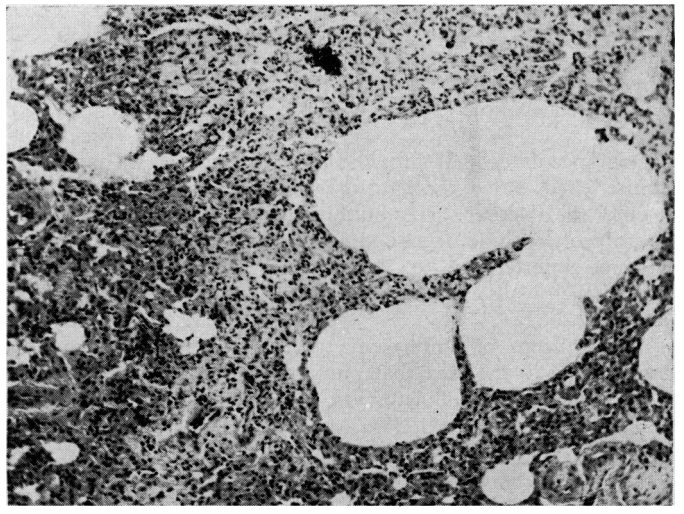

Fig 2B

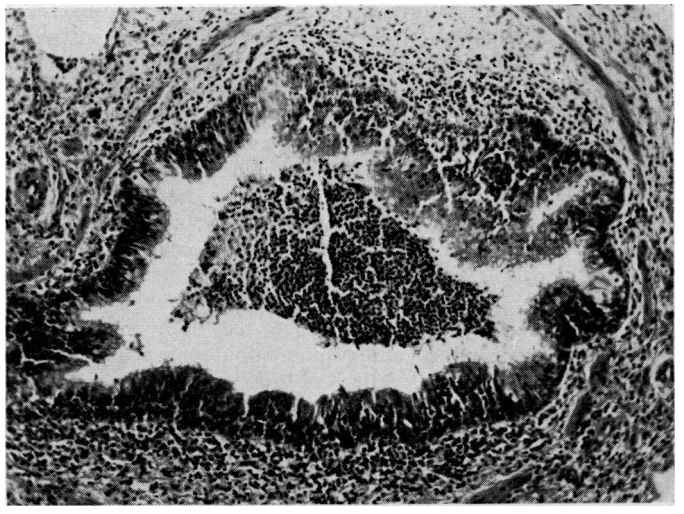

Fig 2C

Fig 2 *Structural emphysema* (Figs 2A, 2B, *and* 2C *are all taken from the same case*)
A, *disruption of alveolar pattern and thinning of alveolar walls. H & E.* ×*13. (Reproduced from Mahaffey, 1962, by kind permission)*
B, *marked fibrosis extending towards the alveoli obliterating the air spaces. H & E.* ×*80*
C, *severe peribronchiolitis, chronic bronchitis, desquamation of epithelium and a plug of mucopus in lumen. H & E.* ×*80*

The findings of Larsson (1936) that emphysematous horses gave positive skin tests with extracts from mouldy hay have not to our knowledge been confirmed.

In human asthma an eosinophilia of the blood is looked upon as confirmatory evidence of allergy, but this guide is not available in the horse, in which eosinophilia is not a characteristic feature of allergic states (Sjoberg & Akerblom 1938). On the evidence presented, one would not be justified in stating dogmatically that equine emphysema is an allergic condition. A great deal more work needs to be done before this point can be settled.

In general, emphysema is a disease of an *individual* animal, but endemic occurrences, with all the horses in a certain stable affected, have been reported (Carlström & Alegren 1940, Morgan 1940).

Pathogenesis

The available evidence would seem to support the theory that, basically, alveolar emphysema in the horse is caused by an acute or chronic spasm of smooth muscle of the bronchioles and alveolar ducts. The onset of this bronchospasm is not dependent upon the presence of an intact vagus nerve (Obel & Schmiterlöw 1948) and therefore must be of either aerogenic or blood-borne origin. Some workers have suggested that emphysema in man can result from primary disease of the

bronchial artery – and the same might be true of the horse. A true arteriosclerosis of the pulmonary circulation has been demonstrated in equine emphysema (Dahme 1960).

One speculates that bronchospasm sets up a diffuse 'check-valve' mechanism which allows air to enter the alveolar spaces but hinders its escape. The alveoli become distended and because there is some interference to the vascular nutrient supply, their walls eventually rupture.

This concept of emphysema in the horse is comparable to the idea that human emphysema is caused by a chronic asthmatic state (Hinshaw & Garland 1956, Lister 1958).

The sharp rise in the incidence of human emphysema since 1945 (*Medical Services Journal* 1962) has focused attention on the similarities between human and equine emphysema. No other domestic animal is naturally subject to generalized alveolar emphysema. McLaughlin *et al.* (1961*a*, *b*) have shown that anatomically the horse is the only domestic animal having lungs comparable to those of man and suggested that the horse is the most proper study animal for the experimental production of emphysema.

If a chronic spasm of the bronchiolar smooth muscle is the root cause of structural emphysema one would expect pathological evidence of the overactivity of this tissue. Hyperplasia of the smooth muscle of the alveolar ducts has been recorded in both human and equine emphysema (Liebow *et al.* 1953, Smith & Jones 1961).

Incidence

Emphysema in the horse is a disease of civilization in that it has never been recorded amongst horses living in the natural state. Of the various factors which affect its incidence management is of prime importance, though it is unusual for diagnostic symptoms to appear before the age of 5 years.

Geographically the disease is widespread, but it is apparently most common in those countries in which horses are stabled for long periods of the year and where the climate is unfavourable to the making of good hay. A fluctuation in the incidence from year to year has been noted in Sweden, where statistics are available owing to the practice of insuring against this disease. In 1939, the owners of 466 horses were indemnified as compared with only 21 in 1943 (Alström & Lauritzson 1953). In Switzerland insurance claims for pulmonary emphysema rise heavily after a year when hay is harvested in poor weather (Udall 1947). In any one year the disease is likely to be at its worst during those periods when the horse is stabled, especially if the ventilation is poor and the atmosphere polluted by dust from hay or straw.

Discussion

There is no evidence to support the oft-quoted contention that hard work or prolonged fast exercise is in itself a cause of emphysema. The young thoroughbred racehorse, for example, rarely exhibits emphysema. This is the more surprising because they are particularly susceptible to infections of the upper respiratory tract and may even be given fast work before being fully recovered. Hug (1937) and Ammann (1939) state categorically that bronchitis is the primary cause of chronic emphysema, whereas Stommer (1887) only found morphological evidence of bronchitis in 2 out of 9 emphysematous lungs.

The laryngeal mucous membrane in the healthy horse is remarkably insensitive. The 'watch dog' cough reflex is so 'sleepy' that it is possible to pass a gastric catheter or even an endotracheal tube through the larynx of a fully conscious horse without necessarily precipitating a single cough and certainly without causing the laryngospasm one would expect in man and most domestic animals. Yet when a chronic laryngitis accompanies a chronic bronchitis the horse coughs as readily as any other animal. In fact the nature of the cough precipitated by gentle external manipulation of the larynx in an emphysematous horse has in the past been regarded as almost pathognomonic of the disease, the cough being prolonged, low, dry and wheezy. It is of some interest to speculate on the significance of the low degree of sensitivity of the normal laryngeal mucous membrane.

A great deal more research is needed before the condition 'broken wind' is fully understood. As already mentioned, the intravenous administration of histamine will produce experimentally the acute clinical condition and will also markedly exacerbate a chronic emphysema that is quiescent. Similarly, such exacerbation may be precipitated by dropping dusty hay in front of a susceptible horse (Alström & Lauritzson 1953). Recently the research team at Davis, California, have been attempting to produce emphysema experimentally by two new methods: (1) By the injection of chlorpromazine into the bronchial artery to produce a generalized endarteritis and partial occlusion (McLaughlin *et al.* 1961*b*, Edwards 1962). (2) By the introduction of small plastic beads into the bronchial artery to achieve the same object (Tyler & Fowler 1963, personal communication). Preliminary reports are encouraging, for an emphysema comparable to the naturally occur-

ring disease in humans and horses has been produced. It would seem logical to conduct investigations into the serology and mycology of emphysema in the horse, e.g. the experimental production of emphysema by inhalation techniques. Mycology would appear to be the key subject to all three conditions under discussion at this meeting.

Acknowledgments: We wish to thank Professor C Formston, of the Royal Veterinary College, London, for his helpful advice; and Dr R K Archer and Mr L W Mahaffey, of the Equine Research Station, Newmarket, for preparation of the pathological material.

REFERENCES

Alström I & Lauritzson I
(1953) *Proc. XV Int. vet. Congr. Stockholm* **1**, Pt. 1, 669; Pt. 2, 318
Ammann K (1939) *Arch. wiss. prakt. Tierheilk.* **74**, 348
Andberg W G, Boyd W L & Code C F
(1941) *J. Amer. vet. med. Ass.* **98**, 285
Arnott W M (1956) *Lancet* ii, 676
Brion, A, Pellerat J & Castric M
(1948) *C. R. Soc. Biol., Paris* **142**, 335
Carlström B & Alegren A
(1940) *Skand. VetTidskr.* **30**, 10
Code C F & Hester H R
(1939) *Amer. J. Physiol.* **127**, 71
Dahme E (1960) *Berl. Münch. tierärztl. Wschr.* **73**, 333
Edwards D W (1962) *Proc. III Aspen Conference on Emphysema* p 581
Fletcher R B (1960) *Vet. Rec.* **72**, 1171
Hinshaw H C & Garland L H
(1956) Diseases of the Chest. London
Hug A (1937) *Schweiz. Arch. Tierheilk.* **79**, 251
Larsson G (1936) *Svensk VetTidskr.* **41**, 408
Liebow A A, Loring W E & Felton W L
(1953) *Amer. J. Path.* **29**, 885
Lister W A (1958) *Lancet* i, 66
McLaughlin R F, Tyler W S & Canada R O
(1961*a*) *J. Amer. med. Ass.* **175**, 694
(1961*b*) *Amer. J. Anat.* **108**, 149
Mahaffey L W (1962) *Vet. Rec.* **74**, 1295
Medical Services Journal, Canada (1962) **18**, 215
Morgan E (1940) *Vet. J.* **96**, 51
Obel N J & Schmiterlöw C G
(1948) *Acta pharm. tox., Kbh.* **4**, 71
Salutini E (1959) *Nuova Vet.* **35**, 101
Sjoberg K & Akerblom E
(1938) *Arch. exp. Path. Pharmak.* **189**, 33
Smith F (1919) History of Veterinary Medicine. London
Smith H A & Jones T C
(1961) Veterinary Pathology. Philadelphia
Stommer O (1887) *Dtsch. Z. Thiermed. Lpz.* **13**, 93
Udall D H (1947) The Practice of Veterinary Medicine. 5th ed. New York
Weiss S, Robb G P & Blumgart H L
(1929) *Amer. Heart J.* **4**, 664
Zanzucchi A (1942) *Nuovo Ercol.* **47**, 269

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Mr Cook's observation that the horse does not possess a cough reflex comparable to the human cough reflex is an observation of outstanding interest. We are so accustomed to regard the cough reflex as the indispensable watchdog of the safety of the respiratory passages that it is difficult to imagine survival without it; the 'impunity quantum', by which I mean the amount of fluid or solid substance which can be inhaled with impunity is in man infinitesimal and must surely be related to the small positive exhaling air pressure which can be used to expel the invader. Beyond that, safety depends upon the cough reflex. Those who operate on the upper respiratory passages, when the watchdog has been put out of action by the anæsthetic, realize the need for permanent vigilance and are aware of the terrible and tragic consequences which can and do occur if this vigilance is allowed to lapse during an operation or is abandoned too soon afterwards.

How can we explain that the horse manages to thrive without this essential protective reflex? I wonder whether the answer is linked to the significant fact that in the horse, through a trachea two or three times the size of the human windpipe, passes a tidal volume twelve times the human one. This quite possibly permits a modulation of expiratory pressures unknown to man and the deployment of a range of positive pressures between the horse's normal expiratory pressure (which is approximately twice the human one) and actual cough. Indeed, it is feasible to regard this mechanism as necessary to obviate coughing when feeding on dry substances. There is no need for a protective cough, because the 'impunity quantum' is relatively great, and, carried by a forceful expiratory airstream, particles sail with ease out of the horse's respiratory tract which according to their weight and size could in man only have been expelled by cough.

Dr J M Bishop spoke on **The Nature of Disturbance of Pulmonary and Circulatory Function in Farmer's Lung.** An account of this paper will be found in *Quarterly Journal of Medicine*, 1963, **32**, 257.

Meeting June 12 1963

The Summer Meeting took the form of a visit to the Central Veterinary Laboratory, Ministry of Agriculture, Fisheries and Food, New Haw, Weybridge, Surrey.